Hypothermia for General and Cardiac Surgery

With Techniques of Some Open Intracardiac Procedures Under Hypothermia

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For many years there have been sporadic attempts to explore the value of lowering body temperature as a therapeutic modality. Recently, interest has been rekindled in the subject because of its potential use as a technique for prolonging the safe period of circulatory occlusion, thus protecting tissue viability during operative intervention in a bloodless field. Of particular appeal was this potential as a technique for operation within the open heart during temporary occlusion of circulation. Enough experience, both laboratory and clinical, has now been accumulated to show that the method of general hypothermia can be applied to man at reasonable risk for a variety of operative purposes. This report is primarily concerned with the technique and risks of hypothermia and with new direct vision cardiac operations, the development of which has been made possible by hypothermia.

PHYSIOLOGIC EFFECTS AND RISKS OF GENERAL HYPOTHERMIA

We define general hypothermia as the physical state of a homothermic individual whose body temperature is below the accepted normal range for that individual. It must not be confused with the word "hibernation," a term used to describe the normal state of an animal which sleeps through the winter. Some such animals, during their winter sleep, do indeed have a profound lowering of body temperature, but other endocrine, metabolic, and physiologic events occur which do not necessarily apply to the hypothermic nonhibernating animal who is cold.

Obviously, many degrees of hypothermia are possible, the range for

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man potentially extending from 37° C. to minus 273° C. Currently, however, we have a body of knowledge and experience in the human with body temperatures in the range of 37° C. to 20° C. only. Within this range, a number of profound changes occur in body physiology. One or more of these effects might be considered desirable in a given patient, either because it might improve some aspect of his physiologic state which is disrupted by his disease, or it might lessen the physiologic stress imposed by the episode of surgery, or it might provide technical opportunities for the surgery itself which could not be or have not been achieved by other means. What are some of these physiologic effects of hypothermia?

Probably the most fundamental change is a progressive fall in the metabolic rate of all the tissues of the body. As measured by oxygen consumption, this fall is essentially linear down to about 25° C., and approximates a 5 per cent decrease for each degree Centigrade which body temperature falls below normal. With the lowered metabolic activity, progressive changes occur in the function of the circulatory, respiratory and nervous systems. The heart rate falls, cardiac output decreases, the blood pressure falls, and a general constriction of the vascular tree occurs. Respirations are steadily depressed and sooner or later stop completely, and, unless artificial respiration is performed, result in profound changes in the metabolic balance of the body. The central nervous system also becomes progressively depressed, resulting in coma and analgesia. The parasympathetic system is suppressed sooner than is the sympathetic system. The effect of hypothermia on endocrine function and on the blood itself is presumed to be significant, but as yet is not understood.

On the basis of these changes, potential clinical applications of hypothermia which might diminish operative risk become obvious. For example, by lowering the metabolic rate, one could combat the hypermetabolism of hyperpyrexia or resistant thyrotoxicosis; by diminishing oxygen need one could return a patient in hypoxia to a state of adequate oxygenation; by reducing heart rate one could diminish the distressing effects of severe tachycardia; or one could achieve prolonged safe regional or general ischemia for technical operative purposes.

If hypothermia were entirely without its own intrinsic risk, there would be little need to debate its utility. However, this is far from the truth, for there are specific lethal risks inherent in the state of hypothermia itself. In our experience with 170 patients as well as with a very large number of experimental animals, the greatest dangers of hypothermia in the range between 20° C. and 30° C. are two: (1) the occurrence of a serious disturbance in the cardiac rhythm, and (2) the development of a hemorrhagic diathesis.

Of these rhythm disturbances, *ventricular fibrillation* is the most lethal. The prevention and successful treatment of this complication com-

prise an essential step in the safe management of the hypothermic individual. Cold itself appears to lower the threshold of the myocardium for fibrillation. The cold heart will fibrillate when subjected to stimuli such as epinephrine, asphyxia, or mechanical trauma, in dose levels which are below the threshold for the warm heart. In our studies of this situation, we have evolved a few techniques which have lowered the incidence and risk of this complication. In the first place, a state of respiratory acidosis being rapidly returned toward normal is a proven cause of fibrillation even in the warm animal. For this reason, we have practiced and strongly advocated the use of artificial hyperventilation throughout the course of hypothermia. This is effective in producing respiratory alkalosis. This practice resulted in a clear-cut diminution of fibrillation in our experimental animals, an effect corroborated in several other laboratories. In addition, we observed that in the normal dog hyperventilation alone caused a reduction in serum potassium. This occurred also in the cold hyperventilated dog. The use of potassium chloride, given by perfusion of the coronary arteries, proved to be of great help in defibrillating the resistant cold heart. Although with an adequate defibrillating instrument this can usually be done by electric shock alone, we have found potassium chloride to be a valuable adjunct in the resuscitation of the cold fibrillating heart.

Interest in the potassium ion led us to investigate some of the agents known to affect the transfer of potassium across the cell membrane. Prime among these was acetylcholine. We determined that the administration of the anticholinesterase neostigmine by coronary perfusion was highly protective against ventricular fibrillation in the cold canine heart. In the last 70 patients, we have used this agent routinely, and the incidence of ventricular fibrillation has been markedly reduced. A similar effect experimentally is obtained by a constant injection of acetylcholine or by continuous stimulation of the distal end of the cut vagus nerve. This evidence suggested to us, and we have proposed the theory, that in the cold state the parasympathetic system is relatively more suppressed or is suppressed earlier than the sympathetic system. Thus, either enhancement of vagal action or suppression of sympathetic action tends to diminish the incidence of ventricular fibrillation occurring during hypothermia.

In summary, by the use of hyperventilation and by coronary perfusion with neostigmine, the incidence of ventricular fibrillation in man in the temperature range 28° C. to 30° C. can be almost eliminated for periods of circulatory occlusion up to eight minutes, even during direct operative intervention on the diseased heart. In patients with normal cardiovascular systems, in whom hypothermia is used for other purposes, the complication in this temperature range is almost nonexistent.

A second important risk of surgery during hypothermia is that of *postoperative hemorrhage*. This may occur from two possible causes. If the

wound is closed during a time when the blood pressure is significantly lowered by the hypothermia, bleeding may occur upon rewarming, with the return of normal pressure. On the other hand, there may be a specific effect of hypothermia upon the blood clotting mechanism. In the dog, circulating platelets are uniformly depressed. This is definitely occasionally true in the human. One of five patients carefully studied in this regard developed a profound thrombocytopenia with subsequent hemorrhage which was fatal 36 hours postoperatively. Other specific effects on the clotting mechanism may also occur. Postoperative hemorrhage has been a serious problem in our hands and we feel that extensive study of the effect of hypothermia upon the blood and upon blood clotting in man is greatly needed to reduce the importance of this complication.

Nonlethal complications include a protracted ileus of the gastrointestinal tract, occasional superficial burns of the skin if diathermy or hot water mattresses are used, and peripheral neuritis if ice water surface cooling is used. The latter complication appears to be a result of the direct injury of peripheral nerves by protracted cold. Immersion longer than 45 minutes in ice water, therefore, is undesirable; after that time the arms and legs should be suspended above the surface of the bath. We have observed no increased incidence of infection and no delay in wound healing due to the hypothermic experience.

MANAGEMENT OF HYPOTHERMIA

Preparation of patients for hypothermic anesthesia is essentially similar to any anesthetic procedure. Morphine, Demerol, barbiturates and scopolamine are given for premedication. Induction is usually with ether. Two intravenous cannulae are placed to assure that this route for fluids or blood will be available. Electrocardiograph needle electrodes are connected and a rectal thermocouple inserted. A surgeon is scrubbed throughout the induction and cooling period, available for cardiac resuscitation if need arise. This precaution was instrumental in saving at least two patients who underwent circulatory arrest before thoracotomy had been performed.

When the patient is in second-plane, third-stage anesthesia, he is placed in a tub of tepid water. The head and arms are held up out of the water. If shivering ensues, curare is given. When vital signs are stable, ice cubes are added to the water. Hyperventilation is deliberately performed throughout the anesthetic experience, except during circulatory occlusion.

The patient is removed from the tub when the rectal temperature has reached a point which is about two-thirds the desired fall. This figure varies somewhat, but the end temperature can usually be estimated in this fashion, within a margin of error of 1° or 1.5° C. To cool an infant requires about 10 to 15 minutes in the tub, while an obese adult may need as long as an hour or an hour and 15 minutes.
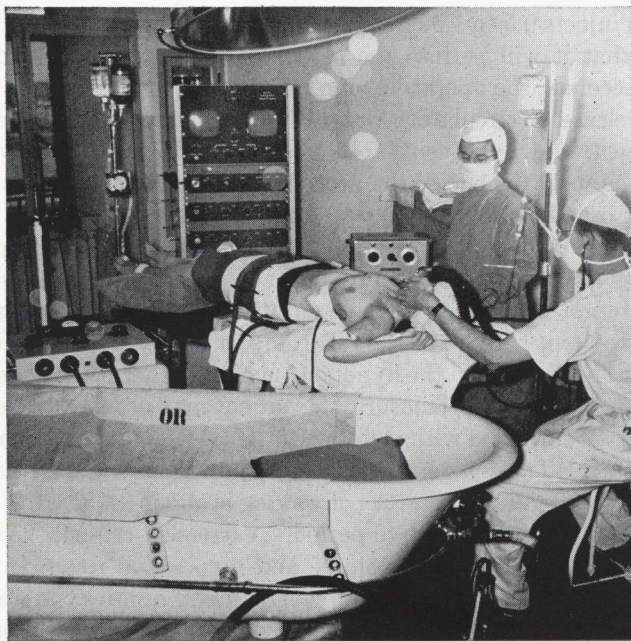
Fig. 371. Operating room scene immediately following repair of atrial septal defect. Equipment included the tub, diathermy unit, rectal thermometer, electronic recording device (ECG and/or pressures), defibrillator, and standard anesthesia machine. A direct writing electrocardiograph and a Beckman pH meter are not shown. (Courtesy of Annals of Surgery, Vol. 142, 1955, published by J. B. Lippincott Co.)

When the patient is removed from the tub, he is thoroughly dried. The pelvic area is wrapped with 1-inch felt, which is taped in place. A standard diathermy coil is then accurately placed, taking care that the patient is supported so that his weight does not lie on the coils. The diathermy is used to counteract a tendency to overdrift in cooling, and to warm the patient immediately following completion of the cardiac procedure. Blood replacement is begun early and an attempt is made to keep pace with the rate of loss. Indeed, transfusion slightly in excess of loss is considered desirable.

About one-half of the patients show auricular fibrillation when they enter the high twenties (°C.). We do not consider this a serious development; most will revert to sinus rhythm at about the same temperature when rewarming.

About five minutes before the moment of circulatory occlusion, further curare is given to prevent contraction of the diaphragm. A determination of blood pH is made at this time. We believe it desirable that the patient be in a state of respiratory alkalosis, with a pH of 7.5 or greater. During occlusion, the lungs are allowed to completely collapse, and respiration is discontinued. The surgeon occludes the inflow of blood to the heart; then after a few seconds occludes the aorta about 1 inch distal to the

valve and injects 0.5 to 1.5 ml. of neostigmine 1:4000 into the base of the aorta so that it will perfuse the coronary system. After an additional 10 or 15 seconds, the operative manipulation is performed.

Upon release of circulatory occlusion, the lungs are again ventilated with oxygen, and hyperventilation resumed. The patient may receive only oxygen until the end of the procedure. If further anesthetic agent is needed, it usually consists of 50–50 nitrous oxide-oxygen.

Diathermy is begun immediately after restoration of circulation, applied intermittently (one minute off and two on) to help prevent skin burns. Attempt is made to achieve an auscultable blood pressure of 90 systolic or above before the thoracotomy is finally closed, in order to avoid later bleeding when the hypotension of hypothermia rises to normal levels. Upon completion of closure, the patient may or may not be further warmed in the tub filled with water at 45° C., depending on temperature. The endotracheal tube is removed when spontaneous respirations appear adequate. The usual temperature of waking is about 34° C.

The immediate postoperative period is extremely critical. Evaluation of effective circulating blood volume and myocardial function is extraordinarily difficult. A few cases of severe shock occurred at this time. Blood volume studies are done at this time to compare with preoperative levels. Improved understanding of the state of the circulation immediately following cooling is badly needed in order to more intelligently control this stage of hypothermia.

HYPOTHERMIA IN SURGERY

Our application of hypothermia has fallen into the following classification of clinical groups: first, noncardiac procedures; second, cardiac procedures without interruption of circulation; and last, open-heart direct-vision procedures during complete cessation of circulation.

Table 1

OPERATIVE PROCEDURES DURING HYPOTHERMIA

(147 patients, 153 procedures)

	PATIENTS	HOSPITAL DAYS
Noncardiac procedures..........................	30	3
Closed cardiac procedures........................	24	7
Open cardiac procedures with circulatory occlusion....	93	15
	147	25

Inspection of Table 1 reveals that 30 patients in this series fell into the group of noncardiac patients. The specific indications for the use of hypothermia were the following: first, the desire to have prolonged periods of regional ischemia during total arterial occlusion. Thus, for

certain neurosurgical procedures including intracranial aneurysm, thrombosis or angioma, temporary occlusion of the carotid and vertebral arteries in the neck allows a bloodless operative field safely for periods up to eight minutes. Such occlusion can be repeated several times, alternating with periods of free blood flow. For thoracic aortic aneurysm, hypothermia protects for periods up to an hour and a half against the spinal cord damage often associated with clamping of the thoracic aorta. The brain damage occasionally occurring during clamping of the carotid artery can be avoided and resection of the artery with anastomosis performed, if necessary, during the resection of a carotid body tumor. During resection of widespread carcinoma in the abdominal or pelvic cavities, the hypotension of hypothermia reduces blood loss. In addition, cross-clamping of the aorta above the renal arteries produces an essentially bloodless field for periods up to two hours with safety. The duration of the operation and the blood loss are much diminished.

The harmful effect of prolonged anesthesia on the severely damaged cirrhotic liver is well known. The use of hypothermia instead of pharmacologic agents for anesthesia in cirrhotic patients undergoing prolonged operation has been very satisfying in a small group of patients undergoing creation of portacaval shunts.

None of the deaths in this group of patients was due to the hypothermia, the deaths occurring many days or weeks postoperatively, of complications of the underlying disease. No patient in this group suffered a serious cardiac arrhythmia.

The diseases for which closed cardiac procedures were performed during hypothermia in 24 patients were of many varieties. Its use was dictated by three main considerations. The first indication was to improve oxygenation in severely cyanotic patients with congenital heart disease. We were favorably impressed by the improvement in color of these children, and feel that the method has value for this purpose. A second indication was to diminish the heart rate. Patients with patent ductus arteriosus, for example, with very large shunts or pulmonary hypertension and with large overactive hearts, came to operation with slow, gently beating hearts. We felt sure they presented better operative risks in this situation, as we feel that prolonged severe tachycardia per se places a great strain on the myocardium. A third indication was the possibility of needing inflow occlusion either to enter the heart directly or to control hemorrhage during the operation. The need did not actually develop in these cases, but we were prepared and could have proceeded if necessary.

In this group, five patients entered ventricular fibrillation, and of these four died. One patient had cardiac standstill, was resuscitated, and is still alive. Three others died during their postoperative periods, of progression of their underlying disease which could not be relieved at operation. Thus, in this group of patients with severe heart disease,

operative manipulation precipitated ventricular fibrillation in five patients. All but one of these occurred in the first portion of the series, before we began the use of neostigmine.

The diseases for which open direct-vision operation was performed within the heart in 93 patients are listed in Table 2. The sole indication in this group was the desire to use total cessation of circulation for a prolonged period in order to achieve a dry operative field within the heart. In our opinion, unobstructed vision is a fundamental principle in

Table 2

DIRECT VISION INTRACARDIAC PROCEDURES WITH CIRCULATORY ARREST
(93 patients, 96 procedures)

DISEASE	PATIENTS	TOTAL CURE	PARTIAL CURE	TOO RECENT	DIED
I. *Pure (Single) Defects*					
Pulmonary valvular stenosis.....	23	11	1	11	0
Pulmonary infundibular stenosis..	2	1	1	0	0
Auricular septal defect (secundum).................	35	21	1	10	3
Auricular septal defect (primum)....................	1	0	1	0	0
Atrio-ventricularis communis....	3	0	1	0	2
Ventricular septal defect........	5	0	1	0	4
Aortic stenosis................	1	0	1	0	0
	70	33	7	21	9
II. *Combined (Multiple) Lesions*					
Tetralogy of Fallot with:					
A. Valvular stenosis..........	8	0	8	0	0
B. Infundibular stenosis......	8	0	5	0	3
Auricular septal defect:					
A. Pulmonary stenosis (trilogy)..............	6	1	3	0	2
B. Ventricular septal defect...	1	0	0	0	1
	23	1	16	0	6
TOTAL....................	93	34	23	21	15

the achievement of sound surgery. The lesions selected for trial were those for which there was, at that time, no adequate method yet developed (atrial and ventricular septal defects), or for which current closed methods yielded poor or inconsistent results (pulmonary stenosis). The method has proved to be extremely valuable for pulmonary stenosis and atrial septal defect of the secundum variety; ventricular septal defects, however, proved technically too difficult to accomplish within the safe limit permitted by this method of hypothermia. On the basis of this experience, we feel that congenital heart lesions that can be repaired through a right heart incision in less than eight minutes of circulatory

occlusion at temperatures not colder than 28° C. can be safely done by direct vision. For pulmonary valvular stenosis and atrial septal defect of the secundum type, open operation can be performed at an operative risk that is as low as any indirect method, and the result obtained is consistently superior.

In this group, nine of the 15 deaths were considered as probably related to hypothermia, while six were apparently unrelated. Intrathoracic hemorrhage immediately postoperatively, often unrecognized until profound shock occurred, was the cause of death in six of these, while ventricular fibrillation was the cause in three. Whether hypothermia per se or the low blood pressure at the time of wound closure was the more important factor in the bleeding patients we cannot say. However, it recently occurred despite our having warmed the patient with diathermy on the table until systolic blood pressure reached 100 mm. of mercury before closing the chest, and despite extrameticulous attention to hemostasis. The patient was found to have a profound thrombocytopenia.

TECHNIQUES OF SOME OPEN INTRACARDIAC PROCEDURES

Pulmonary Stenosis

Pulmonary stenosis may be either valvular or infundibular, and may occur either as an isolated lesion or in association with some other defect of the heart, such as interventricular defect (tetralogy) or interatrial defect (trilogy). The operative technique for the relief of the stenosis is modified primarily by its position in relation to the valve.

1. *Pulmonary Valvular Stenosis.* The patient is supine with the left chest slightly elevated (15 degrees). The incision is a bilateral thoracotomy traversing the sternum and is longer in the third interspace on the left than in the fourth interspace on the right. The sternum is cut in a mortise fashion with two bevels. The pericardium is dissected free of surrounding tissue. An umbilical tape is placed around the superior vena cava extrapericardially between the atrium and the entrance of the azygos vein. A second tape is similarly placed around the inferior vena cava by means of a small incision in the pericardium. Both tapes are brought out under the phrenic nerve to avoid injury to this structure, and threaded through an appropriate piece of rubber tubing. The pericardium is now widely opened across its base and well to the left.

Inspection and palpation confirm the presence of the valvular stenosis. The thickened right ventricular musculature extends entirely to the valve ring, the definite poststenotic dilatation of the pulmonary artery is obvious, and the exact point of origin of the violent systolic thrill in this structure can be felt where the jet through the narrow valve impinges on the vessel wall. The operator moves to the left side of the table; the patient is positioned with a tilt to the right and with elevation of the
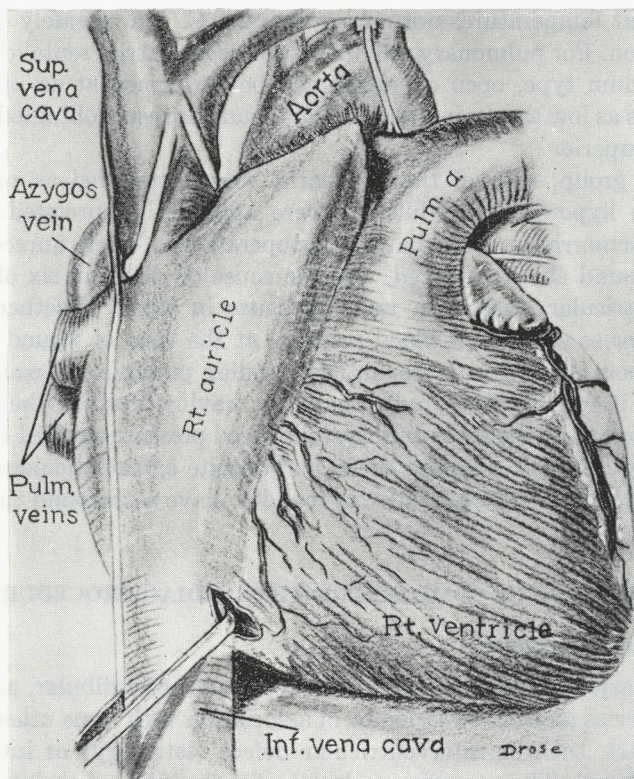
Fig. 372. Artist's depiction of the method of obtaining inflow occlusion to stop the circulation. This method is similar in all intracardiac operations performed during hypothermia.

head of the table, so that the pulmonary artery is the most superior part of the heart. The protocol of the procedure is first completely rehearsed by the operating team. Assurance of ability to rapidly transfuse is confirmed. All is in readiness to proceed.

The adventitia of the pulmonary artery is carefully dissected. Four stay sutures of 5–0 arterial silk are now placed in the vessel for control of the proposed incision. A curved noncrushing Potts clamp is now placed, exteriorizing the appropriate portion of the vessel for an axial incision 3 cm. in length just above the valve ring. The incision is made with the knife and scissors.

Circulatory occlusion is now begun. The venae cavae are completely occluded by advancing the rubber tubing over the tapes. After a few beats of the heart, the aorta is cross-clamped about 1 inch from its emergence. About 0.5 to 1 ml. of 1:4000 neostigmine is slowly injected in the root of the aorta to perfuse the coronary circulation. After a few more beats of the heart, the clamp on the aorta is removed, and a blunt-ended noncrushing auricular clamp is placed across the aorta, snug
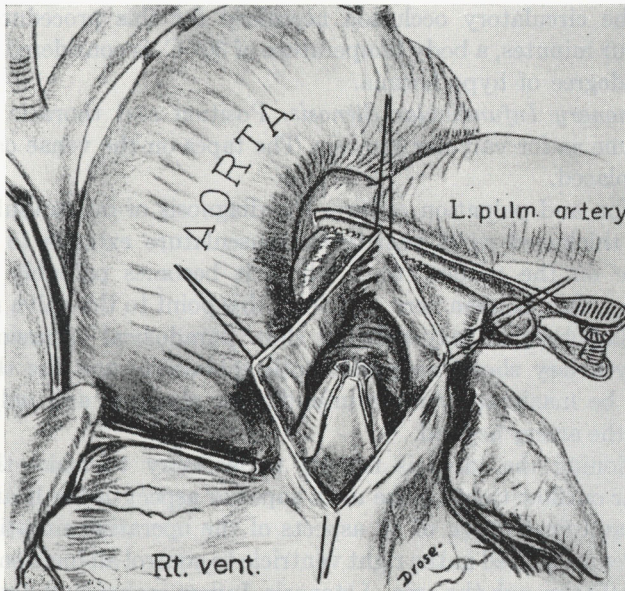
Fig. 373. In repair of pulmonary stenosis, three separate incisions are made in the valve in the line of the undeveloped commissures. These incisions extend all the way to the valve ring. Excision of valve tissue is seldom necessary.

against the myocardium, to occlude the coronary arteries. Another clamp is placed across the pulmonary artery distal to the incision. The curved Potts clamp is now removed (at about one minute of total occlusion), a few beats of the heart empty the right ventricle and the pulmonary valve may be clearly seen in the almost bloodless field.

The valve is grasped with forceps and three cuts in the lines of the undeveloped commissures are made extending all the way to the valve ring (Fig. 373). The finger or an instrument is now passed into the lumen of the ventricle to be certain there is no associated infundibular stenosis.

Withdrawal is now accomplished by loosening the tape around the superior vena cava, thus allowing the right ventricle to fill with blood and expelling all air. The noncrushing curved clamp is now reapplied to the pulmonary artery, closing the incision. Ringer's solution may also be used during this maneuver to be certain all air is expelled from the heart before circulation is resumed. All clamps and tapes are now rapidly removed and circulation is restored.

After a good beat of the heart is re-established, the incision in the pulmonary artery is sutured with interrupted or continuous 5–0 silk sutures. The pericardium is very loosely closed, bilateral chest tubes are placed, and the incision is closed in standard fashion, using wire sutures to the sternum.

Since the circulatory occlusion necessary for this procedure rarely exceeds four minutes, a body temperature of 30° C. is considered to be an adequate degree of hypothermia.

2. *Pulmonary Infundibular Stenosis.* Position and thoracic incision are the same as for valvular stenosis. The tapes on the venae cavae are similarly placed.

Inspection and palpation confirm the diagnosis of infundibular stenosis. The thickened right ventricular musculature extends to a point somewhere on the outflow tract where it becomes palpably thin. A groove may often be seen here, and from this point to the valve ring, the infundibular chamber may actually show paradoxical excursions. The pulmonary artery shows no poststenotic dilatation, and the thrill can be felt to be maximal over the infundibular chamber and only transmitted to the artery beyond.

Repositioning the table is usually unnecessary to make the right ventricular outflow tract be the most superior aspect of the heart. After rehearsal and final check of all aspects of the operative situation, four stay sutures are placed in the right ventricle to control an incision extending vertically through the area of stenosis. Inflow occlusion is now begun. Neostigmine is injected into the clamped root of the aorta. After about 30 seconds, the aortic clamp is replaced by a noncrushing clamp placed across the base of the aorta and the pulmonary artery is also clamped, but about 3 cm. from the valve. The ventricular incision is now made with the knife. After the heart empties itself of blood, the constricting muscle and fibrous tissue are excised with knife and scissors. Palpation of the valve assures no additional stenosis of that structure. The ventricular incision is now closed with a running suture of silk. Just before this suture is completed, the occlusion of the superior vena cava is released, allowing the blood to expel the air from the right ventricle. The suture is rapidly completed, and all vascular occlusions are then released, allowing resumption of blood flow. Additional mattress sutures are placed in the ventricular incision as indicated.

Pericardial and thoracic closure are the same as for valvular stenosis. Since this procedure may take from five to eight minutes, the patient is cooled to about 28° C. in order to achieve certain cerebral protection. When the resection is performed for relief of the stenosis of tetralogy, care must be taken to avoid too complete resection lest the shunt be reversed and lethal strain on the left ventricle be produced.

Table 3 indicates the outcome in 46 patients with pulmonary stenosis. The results obtained by the open relief of valvular stenosis are consistently gratifying and we believe are superior to those achieved by any other method. The merit of the procedure in the treatment of tetralogy with infundibular stenosis remains to be established, although technically the operation is satisfactory.

Table 3

OPERATIONS FOR PULMONARY STENOSIS

DISEASE	PATIENTS	TOTAL CURE	PARTIAL CURE	TOO RECENT	DIED
Valvular stenosis..............	23	11	1	11	0
Infundibular stenosis..........	2	1	1	0	0
Valvular stenosis with atrial septal defect................	5	1	3	0	1
Valvular stenosis with ventricular septal defect...........	8	0	8	0	0
Infundibular stenosis with ventricular septal defect.......	8	0	5	0	3
	46	13	18	11	4

Atrial Septal Defect

The incision for this procedure is placed bilaterally in the fourth interspace and extends further on the right than on the left. The sternum is cut in a "V" shape with double bevel to assure stability after closure. After placing the tapes around the venae cavae, the right pericardium is widely opened, extending the incision across the base and exposing the pulmonary artery.

Confirmation of the diagnosis is made by observation and palpation. The marked enlargement of the right heart chambers and pulmonary vessels is noted. Rarely is pulmonary artery less than double the size of aorta, and a gentle systolic thrill is felt above the valve. With secundum-type lesions, this is the only thrill to be felt. A systolic thrill palpable on the posterolateral aspect of the right auricle indicates regurgitation of an atrioventricular valve, thus strongly suggesting a diagnosis of atrioventricularis communis. Palpation of the defect in the atrial septum can be made by invaginating the appendage, thus giving a rough estimate of the size and location of the lesion. After positioning the table so that the right auricle is the superior portion of the heart, a special noncrushing clamp is applied vertically to the wall of the auricle, pinching up the site of the incision, which is made well posteriorly.

After rehearsal of the proposed manipulation and confirmation of the entire operating room preparedness, traction is made on the caval tapes, thus establishing circulatory occlusion. After about 30 seconds, coronary perfusion of 0.8 to 1.5 ml. of 1:4000 neostigmine is performed. A noncrushing clamp is then placed across the base of both great vessels from the left side. The auricular clamp is now removed, allowing entrance into the right auricle. The mitral valve is palpated through the defect to assure its adequate size. Residual blood is sucked out. The clearly visible defect, tented up by the traction of the caval tapes, can now be easily sutured. We usually place a mattress suture of 00 silk at the lower end,

Fig. 374. *Upper,* Photograph at operation of closure of secundum-type atrial
septal defect. A mattress suture is first placed at each end of the oval defect. *Lower,*
The upper mattress suture is run as a continuous suture, completely closing the
defect. The two sutures are tied at the lower end of the closure. (Courtesy of Surgery,
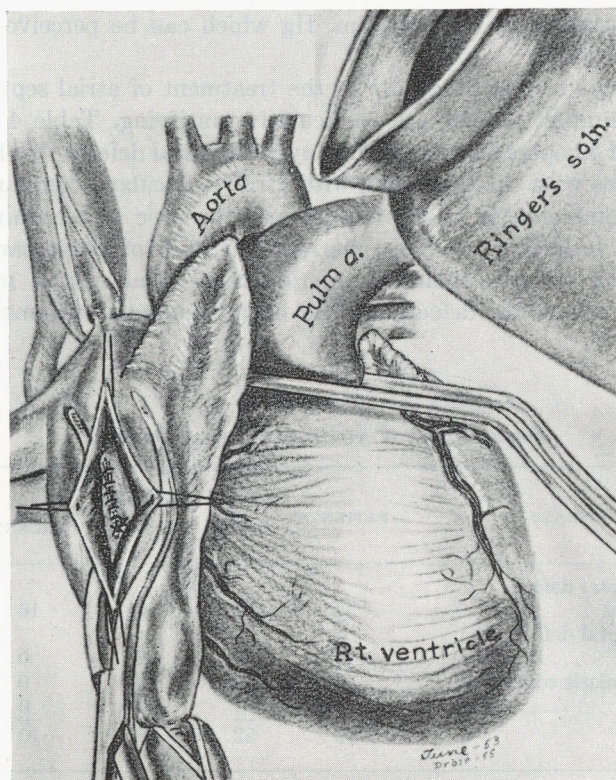Vol. 38, 1955, published by C. V. Mosby Co.)

Fig. 375. Escape from the heart requires removal of all air to avoid air embolism. The right auricle is filled with blood, by releasing the superior vena cava, and by Ringer's solution poured into the incision, which is finally clamped under the surface of the solution.

leaving one end long. A second mattress suture is placed at the upper end and is now run as a continuous suture downward toward the first. Just before tightening the last throw of the suture, an instrument is placed through the defect into the left auricle, and the auricle is filled with Ringer's solution, thus allowing all air to escape from the left heart. The sutures are then tied to each other under the fluid level (see Fig. 374).

Retreat from the heart is achieved by holding up the edges of the incision and releasing the traction on the superior vena cava. As the blood entering the heart fills the auricle, the noncrushing clamp is again applied. All vascular occlusions are now rapidly released. After an adequate circulation has been resumed, the auricular incision is closed with interrupted mattress sutures of silk.

Closure of the pericardium and thoracic wall is conducted as described above, after bilateral tubes have been placed. The chest is not actually closed until rewarming with diathermy has resulted in a systolic

blood pressure of at least 90 mm. Hg which can be perceived by the anesthesiologist.

The results of this procedure in the treatment of atrial septal defect of the secundum variety are particularly gratifying. Table 4 lists the outcome of 42 operations for various types of atrial defects. We have had poor success with three patients with atrioventricularis communis. The uniform completeness of the closure and the ease of recognizing and managing variations, such as aberrant entrance of pulmonary veins, multiple defects, or prominent inferior vena caval valves, make the closure of secundum defects by the open technique extremely gratifying.

Table 4

OPERATIONS FOR AURICULAR SEPTAL DEFECT

DISEASE	PATIENTS	TOTAL CURE	PARTIAL CURE	TOO RECENT	DIED
Auricular septal defect (secundum)..................	36	21	1	10	4
Auricular septal defect (primum)..................	1	0	1	0	0
Atrioventricularis communis.....	3	0	1	0	2
Trilogy......................	2	1	0	0	1
	42	22	3	10	7

SUMMARY

1. General hypothermia can safely be utilized for the solution of a variety of surgical therapeutic problems.

2. For certain vascular and neurosurgical procedures, prolonged regional ischemia is made tolerable.

3. In cyanotic heart disease or in patients with severe tachycardia, the operative risk of standard procedures can be diminished.

4. By permitting relatively safe total circulatory occlusion for periods up to eight minutes, general hypothermia in the range of 28° to 30° C. has become an established technique for direct vision intracardiac procedures.

5. The techniques for the relief of pulmonary stenosis and the closure of atrial septal defects are described. These methods have given unusually gratifying results at low operative risk.

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